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Influence of Feed on Onset of Disease in Subacute Myelo-Optico-Neuropathy Virus-Infected Mice

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An enriched feed, specially prepared to affect reproduction of mice, significantly suppressed the onset of disease in subacute myelo-optico-neuropathy virus-infected mice, whereas low-calorie feed enhanced the incidence of disease.

Subacute myelo-optico-neuropathy (SMON) virus, a new human herpesvirus, has been isolated from human cases of SMON and found to be a variant of avian infectious laryngotracheitis virus (1, 3). We reported also that C57BL/6 newborn mice are susceptible to SMON herpesvirus, but susceptibility of the mice to the virus varies considerably depending upon the individual litter of mice (2). The reason for different results in studies of SMON virus-infected C57BL/6 newborn mice has continuously been investigated. The purpose of the present communication is to report the fact that the onset of disease in SMON virus-infected mice is influenced by the kinds of feed given to the dams.

TABLE 1. *Composition of pellet feed for mice*[a]

Feed	% of:					Cal/g
	Water	Crude protein	Crude lipid	Crude ash	Crude fiber	
CMF	7	28.1	9.1	6.6	3.8	3.8
MF	7	25.0	5.0	7.4	4.2	3.5
LF	7	20.4	3.9	6.9	7.2	3.4

[a] More detailed information on feed composition can be obtained from the Oriental-Kobo Co., Ltd., 3-6-10, Azusawa, Itabashi-Ku, Tokyo 174, Japan.

SMON herpesvirus of the Watanabe strain, which had been propagated in human diploid cells, was used in this work. The virus was inoculated intracerebrally or intraperitoneally into C57BL/6 newborn mice at birth. Only one kind of pellet feed, "MF," as indicated in Table 1 summarizing the compositions of three kinds of pellet feed, was supplied to pregnant mice in a previous study (2). However, it was demonstrated in the present study that a specially prepared feed to affect reproduction of mice "CMF," given to the dams had an influence on the onset of disease; namely, the incidence of disease in SMON virus-infected newborn mice whose dams consistently received CMF before and after delivery was compared with that in SMON virus-infected newborn mice whose dams were fed "LF," instead of CMF, after delivery. The incidence of disease was also examined in SMON virus-infected newborn mice whose dams were given only LF throughout the pre- and postpartum periods.

TABLE 2. *Influence of feed on onset of disease in SMON virus-infected mice*

Feed (before and after delivery)	Virus infected[a] (TCD_{50})	Route of inoculation[b]	Onset of disease[c] (per litter)	(Total)
CMF-CMF	$10^{2.6}$	i.c.	0/3, 1/7, 0/6, 0/7, 0/4	(1/27)
		i.p.	0/10, 0/3, 0/5, 0/6,	(0/24)
CMF-LF	$10^{2.6}$	i.c.	1/5, 3/4, 0/4, 2/6, 2/9	(8/28)
		i.p.	4/8, 0/3, 1/5, 0/3,	(5/19)
LF-LF	$10^{2.6}$	i.c.	5/6, 1/4, 6/6, 1/4, 5/9	(18/29)
CMF-LF	Control	i.c.	0/6, 0/4, 0/7, 0/6	(0/23)
LF-LF	Control	i.c.	0/4, 0/6, 0/3, 0/5	(0/18)

[a] Virus was inoculated at birth. TCD_{50}, 50% tissue culture infective dose.
[b] i.c., Intracerebral; i.p., intraperitoneal.
[c] Diseased mice/mice observed. Mice that died within 1 week were excluded from the results.

The results (Table 2) revealed that the inci-

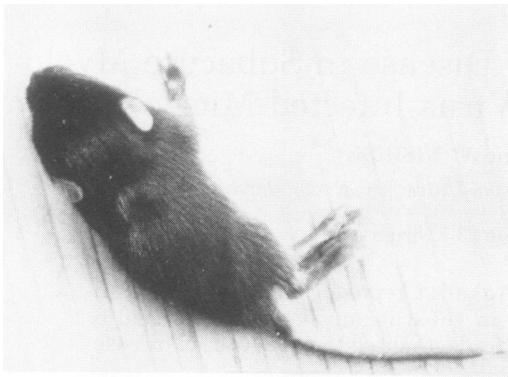

FIG. 1. *Diseased mouse showing typical symptoms due to SMON virus infection.*

dence of disease in the CMF-CMF group was extremely low, and no mouse showed development of disease after intraperitoneal inoculation of the virus, whereas the incidence in the CMF-LF group was nearly the same as that in mice used for the previous study and disease developed in some mice after intraperitoneal inoculation. The LF-LF group was likely to be most susceptible to the virus. After 2 to 3 weeks or more of incubation, in the animals who showed the onset of disease, a loss of weight, piloerection, and characteristic paralysis of hind limbs were manifested, resulting in either death or recovery (Fig. 1). These symptoms were transient in some mice, so that mice should be observed every day, starting 10 days after inoculation of the virus, for a period of 2 months. The main pathological findings were symmetrical axonal degeneration and demyelination of the spinal cord, without inflammatory change (2).

It is reasonable to presume that feed can possibly influence the immune response of hosts, which may regulate the onset of disease, since it was shown previously that mice that showed no signs of infection had a lower virus titer in the brain and a higher antibody titer (2). This finding concerning diet is of practical significance because SMON virus infection in humans has recently been shown to exist also in countries other than Japan (unpublished data).

LITERATURE CITED

1. **Inoue, Y. K.** 1975. An avian-related new herpesvirus infection in man—subacute myelo-optico-neuropathy (SMON). Prog. Med. Virol. 21:35–42.
2. **Nakamura, Y., and Y. K. Inoue.** 1972. Pathogenicity of virus associated with subacute myelo-optico-neuropathy. Lancet i:223–226.
3. **Nishibe, Y., and Y. K. Inoue.** 1974. Relationship between SMON virus and avian infectious laryngotracheitis virus. Arch. Gesamte Virusforsch. 45:278–284.